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Minireview

The assessment of doses and effects from intakes of radioactive particles

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ABSTRACT

The behaviour of radionuclides after entry into the body and the radiation doses received by individual tissues depend on the chemical nature of the element, the physicochemical form of the intake, the radioactive half-life of the isotope and the type and energy of the emissions. Ingestion of radionuclides in insoluble particles will result in radiation doses being delivered to tissues of the gastrointestinal (GI) tract; other tissues will also be irradiated by nuclides with penetrating photons emissions (gamma) but doses may be largely confined to the GI tract for charged particle emissions (alpha, beta). Ingestion of more soluble forms will lead to greater absorption to blood and deposition in other tissues and therefore may result in greater doses to other tissues. Similar considerations apply to inhaled material and to the entry of radionuclides through cuts or wounds. For ingested materials, including particles, more information is needed on uptake and retention in intestinal tissues and consequent doses to sensitive cells, particularly for alpha emitters. There has been concern that the pattern of distribution of activity throughout irradiated tissues may influence the extent of damage, particularly for alpha emitters because of the localised deposition of energy and their greater relative biological effectiveness compared with beta/gamma emitters. Aggregation of activity has the potential to result in greater acute tissue damage and has been shown, for example, to result in focalised pneumonitis and fibrosis in the lung and ulceration of the skin. The main long-term effect of irradiation of tissues is the induction of malignant change, although hereditary disease may also be of concern following irradiation of the gonads. There are only limited data available to compare the effect on cancer induction of heterogenous and homogeneous irradiation of tissues. However, the available information, for irradiation of the lung, skin or liver, indicates that in general nonuniform alpha irradiation from radioactive particles is no more hazardous, and may be less hazardous, than if the same activity is uniformly distributed.

Key words: Radionuclides; radiation effects.

INTRODUCTION

Radiological protection criteria needed for establishing limits on intake for radionuclides and for assessing their effects depend upon an understanding of their behaviour in the body to allow the calculation of tissue doses, the identification of tissues at risk, and a knowledge of dose-response relationships for individual tissues following uptake of β/γ and α emitters. The incorporation of radionuclides into insoluble particles can result in substantial changes in the distribution of radionuclides in the body after intakes either by ingestion or inhalation, compared with that found after intakes of soluble forms, with consequent changes in the distribution of radiation doses between tissues and subsequent effects. Additionally there has been concern that the pattern of energy deposition throughout irradiated tissue may also influence the degree of damage. This has been considered principally in relation to intakes of α

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emitters, for which radiation damage can be much more localised.

This paper will consider first how radionuclide intake in insoluble particles may alter radiation doses to tissues, with emphasis on intake by ingestion. It will then examine the extent to which incorporation of radionuclides into particles may influence radiation damage. Finally it will review dosimetric models for assessing doses from ingested radionuclides, having regard for the physicochemical form(s) of the intake.

INTAKE OF RADIOACTIVE PARTICLES

The behaviour of radionuclides after entry into the body by either ingestion or inhalation is substantially influenced by the physical and chemical form of the intake. When radionuclides are taken in as relatively soluble forms their uptake into body tissues and subsequent distribution and excretion will depend largely upon the affinity of the element for the various transport systems in the body. Thus ^{137}Cs, which is present in weapons fallout, behaves much like potassium. It is readily absorbed either from the gastrointestinal (GI) tract or the lungs and is subsequently taken up by most tissues. As a consequence it gives a similar radiation dose to all tissues. In contrast, ^{90}Sr behaves like calcium, with about a third of the activity ingested in food being absorbed into the blood and with much of this activity depositing in the skeleton which consequently receives the highest radiation dose. If these radionuclides are incorporated into insoluble radioactive particles, however, only a small fraction of the ingested activity may enter the blood and the overall dose to the tissues of the body is very much reduced. This reflects the short transit time of radionuclides in the GI tract and the longer retention times of ^{137}Cs and ^{90}Sr in the rest of the body. Similar results have been obtained with ^{239}Pu. Although absorption of this α-emitting radionuclide is generally low it can be present in a wide range of chemical forms which have very different absorption characteristics in the gut. When present in soluble complexes in food, levels of absorption up to about 0.1% (10^{-3}) can be obtained but for insoluble plutonium dioxide uptake from the gut may be less than 10^{-5} (ICRP, 1986). Plutonium is retained in the body principally in the skeleton and liver with half-times of retention of tens of years. The dose to the whole body therefore depends mainly on the amount of ^{239}Pu entering the blood. The effective dose (see later) for an f_1 (gut transfer factor) of 10^{-3} is about 50 times that for an f_1 of 10^{-5}. Similar considerations apply to the absorption of various physicochemical forms of radionuclides from the lungs following their inhalation.

These examples illustrate the importance of physicochemical form in determining the radiation dose from an intake of a radionuclide and for predicting its likely effects.

DOSES TO TISSUES

In addition to the effect of incorporation of radionuclides into particles on the distribution of dose between tissues, there has been concern that the pattern of energy deposition throughout irradiated tissue may influence the extent of damage. This has arisen principally in the context of exposure to α emitting radionuclides as radiation damage will be much more localised than for β/γ emitters. Alpha emitters also have a much greater relative biological effectiveness (RBE; see later). Deposition in the human lungs (mass 1 kg) of 1 kBq of ^{239}Pu as particles with an activity median aerodynamic diameter (AMAD) of 0.1 μm, for example, will result in irradiation of about 50% of the lung volume. For the same amount of activity inhaled as 1 μm particles, however, only about 0.05% of the lung will be irradiated, but to much higher doses. Concentration of activity in tissues, with consequent increases in local doses has been shown to result in greater acute tissue damage in the vicinity of the particle(s), causing fibrosis and ulceration. In severe cases this can result in loss of tissue function and even death. The main long term effect of irradiation at low doses is, however, the induction of malignant change, although hereditary disease may also be induced through irradiation of the gonads.

There are a limited number of studies that have directly compared the effects of homogeneous irradiation of tissues with heterogeneous irradiation from 'hot' particles. Much of the available data has been reviewed by Task Groups of the International Commission on Radiological Protection. A review of the biological effects of inhaled radionuclides concluded that some studies suggested that the lung cancer risk associated with inhaled plutonium particles may be slightly greater per unit dose than the risk for more soluble and hence more uniformly distributed activity. Other studies designed specifically to test the hot particle hypothesis had been negative. There was no suggestion that hot particles could give a substantially higher risk than the same activity uniformly distributed throughout the lung (ICRP, 1980). In a

review of radiation damage to the skin it was concluded that, for the induction of cancer, uniform exposure of the skin was more carcinogenic than nonuniform irradiation (ICRP, 1991). For deterministic effects, however, in particular acute ulceration, local doses from particles are the main concern. A study by Brooks et al. (1969) compared the effects of α particle irradiation from ^{239}Pu on the induction of chromosome aberrations in the liver of the Chinese hamster after administration of ^{239}Pu citrate, which gives a more uniform distribution of dose in tissues, and $^{239}PuO_2$ particles. The more uniform distribution of activity produced more chromosome aberrations and it was therefore likely that long term risks of liver cancer from the ^{239}Pu particles were likely to be less than from a uniform distribution of activity.

It may be concluded that a uniform distribution of activity is likely to be more carcinogenic than a nonuniform distribution. For the calculation of tissue doses to provide a basis for assessing long term health effects, the calculation of average tissue dose is therefore likely to be conservative. This is the approach that the ICRP continues to use (ICRP, 1994). For assessing the risk of deterministic effects, however, it may be necessary to calculate local doses.

DOSIMETRIC MODELS FOR THE GI TRACT

Doses to the GI tract from an ingested radionuclide depend on the type and energy of emissions, radioactive half-life, the proportion absorbed and the route of excretion from the systemic circulation. In addition, transit times vary between individuals and with age. Retention of nuclides in the wall of the GI tract may be important, particularly in newborn infants. The absorption of radionuclides from the GI tract will depend on the chemical nature of the element concerned and the form in which it is ingested. In all cases where data are available to make comparisons, absorption is greater after ingestion of inorganic forms in solution than after ingestion of radioactive particles.

Current ICRP model for the GI tract

In 1966, Eve reviewed data on the transit of materials through the GI tract and other parameters necessary to calculate doses. Transit times were based largely on information from clinical studies using barium meals but also included reference to studies using materials labelled with iron-59 or lanthanum-140. These data provided the basis for the dosimetric model of the GI tract in ICRP Publication 30 (1979). It is a model with

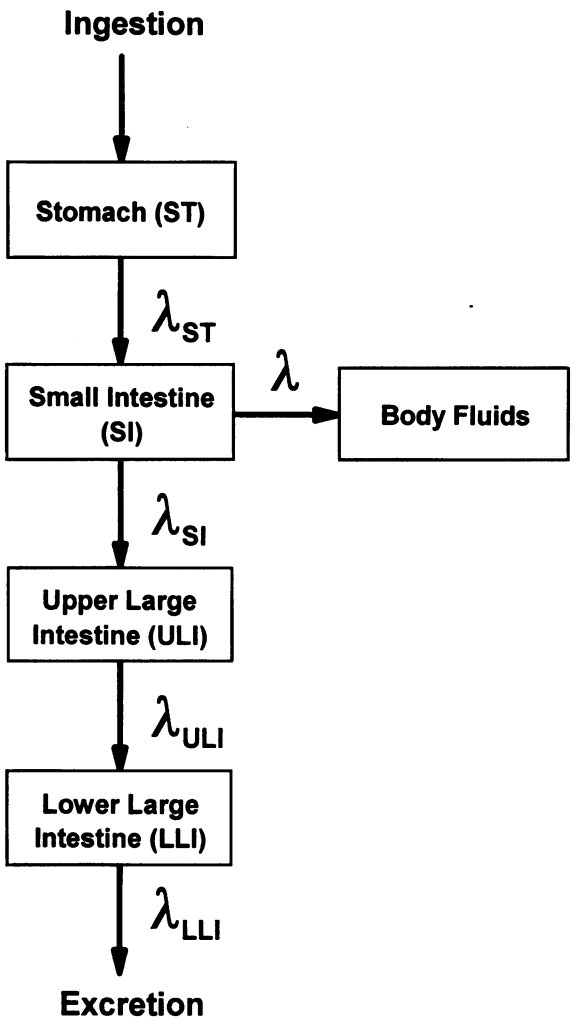

Fig. 1. ICRP Publication 30 (1979) model of the GI tract.

4 linked compartments: the stomach, small intestine, upper large intestine and lower large intestine (Fig. 1). The model allows for time-varying input to the stomach and one-way intercompartmental transfer with 1st order kinetics. The absorption of materials to blood is taken to occur in the small intestine. The transfer rate coefficients for movement of intestinal contents are equal to the reciprocal of the mean residence times, taken to be 1 h for the stomach, 4 h for the small intestine, 13 h for the upper large intestine and 24 h for the lower large intestine on the basis of observed ranges of 25–120 min, 1–7 h, 6–22 h and 15–72 h, respectively.

Doses are calculated separately for the mucosal layer of each region of the GI tract. For penetrating radiations, the average dose to the wall of each region is used as a measure of the dose to the mucosal layer. For nonpenetrating radiations, the fraction absorbed by the mucosal layer is taken to be equal to 0.5 v/M where M is the mass of the contents of that section of the GI tract and v is a factor between 0 and 1

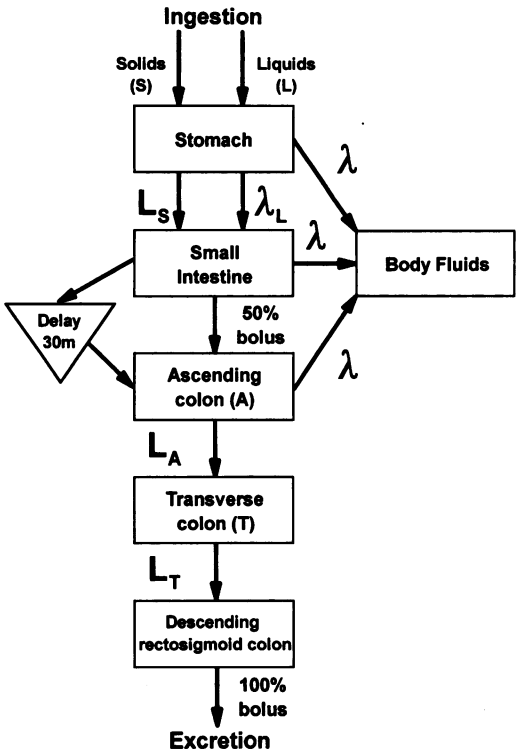

Fig. 2. Stubbs (1992) model of the GI tract.

representing the proportion of energy reaching sensitive cells. The factor of 0.5 is introduced because the dose at the surface of the contents will be approximately half that within the contents for non-penetrating radiations. For β particles, v is taken to be 1. For α particles, a value of 0.01 is used. This value of 0.01 is arbitrary, the experimental evidence that it is warranted being from an acute toxicity study in rats in which the LD_{50} for ingested yttrium-91 was estimated as about 12 Gy while a dose to the mucosal surface of more than 100 times greater from ^{239}Pu had no effect (Sullivan et al. 1960).

New models for the GI tract

A new model of the GI tract is required for 4 main reasons. (1) ICRP have recently recommended specific risk estimates for radiation-induced cancer of the oesophagus, stomach and colon. (2) There are good new data on gut transit of materials, obtained using noninvasive scintigraphic procedures. (3) Doses from radionuclides retained in intestinal tissue need to be considered. (4) Uncertainty in doses to sensitive cells, particularly from alpha-emitters, requires further attention.

The ICRP have recently published revised estimates of radiation risks, based largely on reassessments of the incidence of cancer in the survivors of the atomic bombs at Hiroshima and Nagasaki (ICRP, 1991). Specific risk estimates for cancer of the oesophagus, stomach and colon are now included and the overall estimate of fatal cancer risk has increased from 0.0125 Sv^{-1} in the 1977 recommendations to 0.05 Sv^{-1} for the general public in the 1990 recommendations (ICRP 1977, 1991). This new risk estimate means that, for example, if 2000 people were all exposed to 0.01 Sv, 1 additional cancer would be expected compared with a similar unexposed group.

Recently, a revised GI transit model has been developed (Fig. 2) on the basis of a substantial body of new data obtained by noninvasive scintigraphic procedures (Stubbs, 1992). These techniques have the advantage of avoiding any possible disturbance of normal GI motility. Currently, transit through the oesophagus is not included but this will be considered in developing the model for ICRP (Stubbs, 1992). Removal from the stomach to the small intestine is described by a combination of zero and 1st order kinetics. Because liquid emptying is a 1st order process, the gastric emptying of liquids is best modelled by an exponential function of time. In contrast, the emptying of solids is thought to be a linear or zero-order process. Gastric emptying half-times were found to vary over a wide range; the values adopted in the model were 30 min for liquids and 60 min for solids. Transit through the small intestine is taken to be independent of the gastric emptying rates. A transit time of 4 h was assumed. Upon filling of the terminal ileum (gastric emptying time plus SI transit time of 4 h), the model assumes that 50% of the activity is transferred to the ascending colon in a single bolus and that the remaining activity is transferred 30 min later as a bolus. The model partitions the large intestine into the ascending colon (AC), transverse colon (TC) and the descending/rectosigmoid colon (DRC). The activity transferred to the AC is assumed to be instantaneously distributed throughout the contents and completely retained during a 30 min caecal filling time interval. The transit times through the AC and TC were taken to be 8.4 h and 7.3 h, respectively. Elimination of activity from the GI tract was assumed to occur at 15.9 h after complete emptying of the transverse colonic contents to the descending colon. The model allows for absorption to blood from the stomach, small intestine and ascending colon.

The differences between the ICRP (1979) model of the GI tract and the Stubbs (1992) model have not yet been fully assessed. However, it would appear that the effect on mean residence times and doses to the different regions will not be large. For the example of the radionuclide, ^{99m}Tc, ingested in a nonabsorbable

Table 1. *Intestinal retention of ^{238}Pu in newborn animals*

Species	Age at ingestion (d)	Time after ingestion (d)	Ingested activity (%)* 1	2	3
Rat	6	5	0.03	5.5	16.7
	12	5	0.01	4.6	36.3
Guinea pig	6	5	0.006	0.007	0.009
	12	5	0.008	0.009	0.009

* 1, 2 and 3 refer to 3 equal lengths of small intestine.

form such as sulphur colloid, the largest differences in residence time would be for the small intestine with a 44% increase in the revised model. The Stubbs model and one being developed by Nosske & Simko (personal communication) will form the basis for a revised ICRP model.

High levels of intestinal retention of radionuclides have been observed in the immediate postnatal period in some mammalian species, associated with high levels of absorption to blood (Fritsch et al. 1988; NEA, 1988; ICRP, 1989). The levels of intestinal retention in different species appear to be related to the extent of pinocytotic activity. Table 1 compares the retention of ^{238}Pu in the small intestine of rats and guinea pigs given ^{238}Pu nitrate (Harrison & Fritsch, 1992). Autoradiographic studies show that for rats (Fig. 3*a*), as for pigs, the high levels of retention are confined mainly to the epithelial cells (Fritsch et al. 1988; ICRP, 1989). The kinetics of loss have been considered as a dynamic process involving the normal migration and sloughing of epithelial cells from the tips of villi. In guinea pigs (Fig. 3*b*), baboons and macaques, low levels of Pu are retained mainly in macrophages in the lacteal region in the tips of the villi (Fritsch et al. 1988; ICRP, 1989). Studies in which ^{239}Pu was administered to newborn rats and guinea pigs as oxide particles showed lower levels of retention than for soluble forms. In rats given ^{239}Pu oxide at 2 d of age, total retention 7 d later accounted for 0.025–0.5% of the administered amount. In guinea pigs, most retained activity was adsorbed onto the surfaces of villi (Fig. 4). No association with Peyer's patches was observed in neonatal or older animals. These data and evidence on the intestinal persorption of particles will be taken into account in revising the ICRP model of the GI tract.

Estimating the dose to sensitive cells in the intestine from short range radioactive emissions is inherently uncertain. The arbitrary assumptions made for alpha emitters in the current model are summarised above (ICRP model). Until recently there have been no good animal models with which to pursue this problem experimentally. The *Min* (multiple intestinal neoplasia) mouse, genetically heterozygous for the *Apc* tumour suppressor gene, potentially offers such an animal system and is currently being used to study chemical carcinogenesis in the intestinal tract (Moser et al. 1990, 1993). Germ-line mutations in the human adenomatous polyposis coli (*APC*) gene result in familial adenomatous polyposis (FAP), an autosomal dominantly inherited disease characterised by the early onset of multiple adenomatous polyps of the large intestine with a high likelihood of developing colorectal cancer. The phenotypic and genotypic similarities between murine *Min* and human FAP suggest that the *Min* mouse is a good model for the human disease. Furthermore, because mutation of the *Apc* gene occurs somatically in the development of sporadic colorectal tumours in man, *Min* mice are also a suitable model for colorectal cancer in general.

Absorption to blood

In the ICRP (1979) model of the GI tract, the absorption of radionuclides is assumed to occur instantaneously from the small intestine. The level of absorption varies widely between different elements, depending on their chemical nature, with virtually complete absorption of iodine and caesium, for example, and less than 0.1% for the actinide elements, plutonium and americium (fractional absorption or f_1 of < 0.001). The chemical form of an ingested element might affect the absorption of some elements with the general observation that incorporation into food may lead to greater absorption than ingestion of inorganic forms of an element. There is good evidence from animal experiments, with supporting human data for some elements, that the absorption of a large number of elements is greater in newborn mammals than in adults. Absorption values recommended by ICRP for members of the public are shown in Table 2 (ICRP, 1989, 1993, 1995).

In all cases where information is available on ingested radioactive particles, the absorption of radionuclides is lower than for soluble inorganic forms. For example, absorption of ^{239}Pu after ingestion of oxide particles has been shown to be at least an order of magnitude lower than after administration as the nitrate or citrate (ICRP, 1986). Talbot (1991) measured absorption of ^{137}Cs from irradiated reactor fuel particles (2–10 μm) in adult rats to be less than 0.1 compared with the ICRP value of 1 for Cs as soluble inorganic forms (ICRP, 1979) or in food (ICRP, 1989).

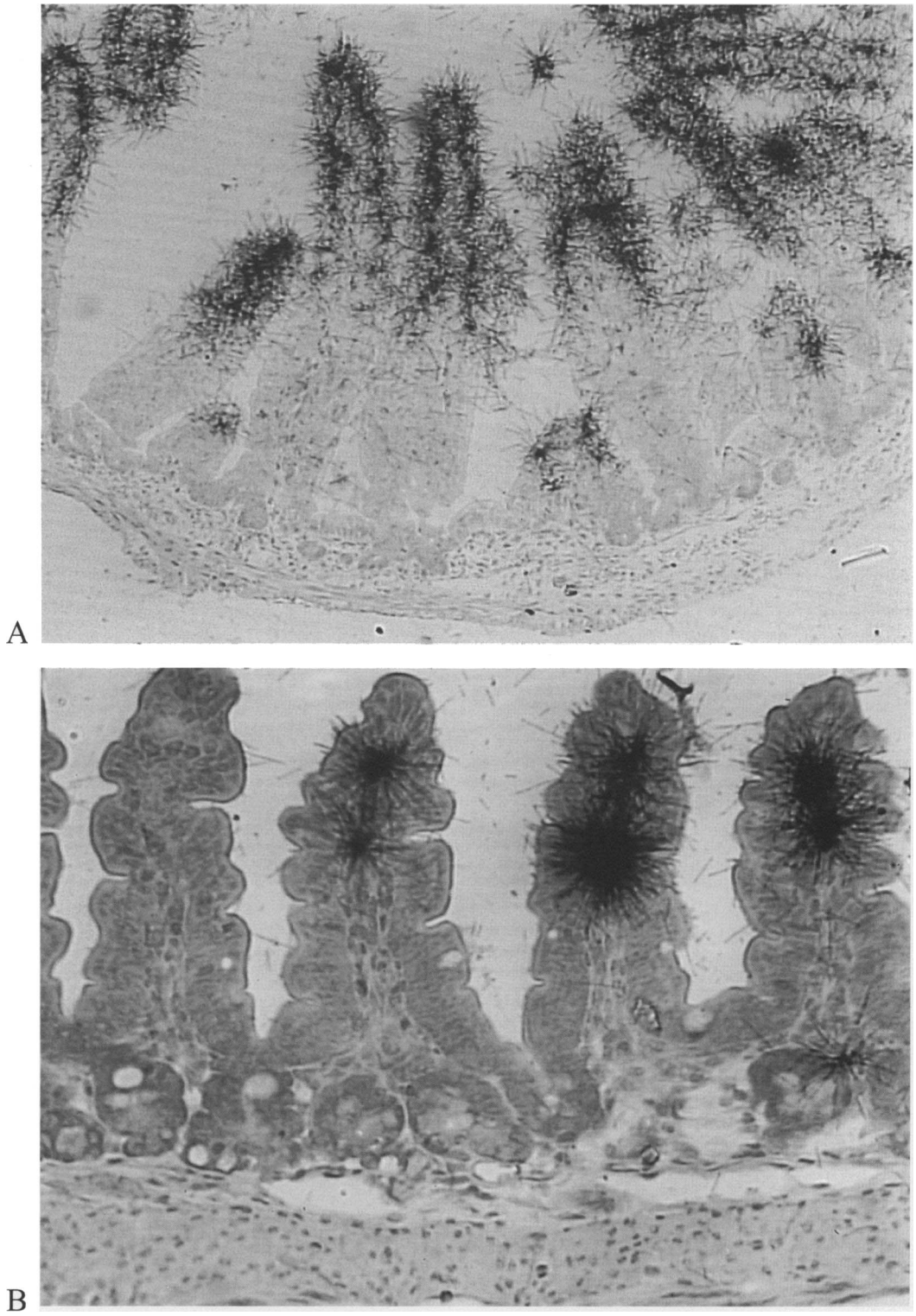

Fig. 3. Autoradiographs showing the retention of ^{238}Pu in the villi of the small intestine of newborn rats (A) and guinea pigs (B) after administration as the nitrate.

Doses from ingested radionuclides

For ingested radionuclides, doses are currently calculated using the GI tract model discussed above and element specific models for the tissue uptake and retention yof radionuclides reaching blood (ICRP, 1979, 1989). To provide a tool for the interpretation of absorbed dose in different organs in terms of the total risk of cancer and hereditary effects, ICRP use the concepts of equivalent dose and effective dose (ICRP, 1977, 1991). Radiation weighting factors take account of the relative biological effectiveness of different

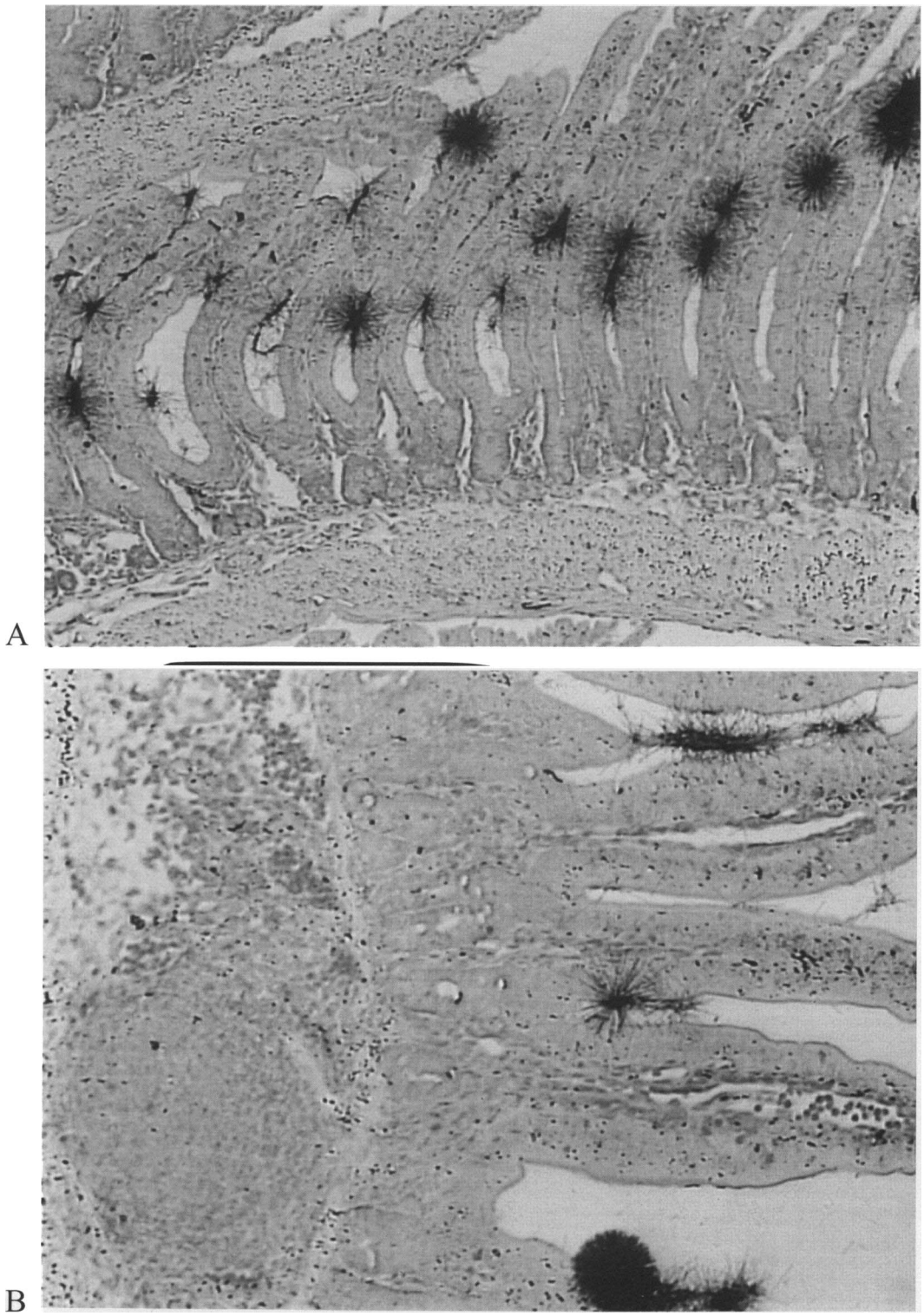

Fig. 4. Autoradiographs showing the retention of ^{239}Pu on the villi of the small intestine of newborn guinea pigs after administration as oxide particles ($\sim$ 1 μm).

radiation types in causing malignancy or genetic damage. Thus the absorbed dose in Gy (Joules kg^{-1}) is multiplied by a radiation weighting factor of 20 for alpha irradiation and 1 for beta and gamma radiations to give the equivalent dose in sieverts (Sv). Tissue doses are commonly integrated over a 50 y period for adults or to age 70 y for children and the resulting values are referred to as committed equivalent doses. Tissue weighting factors are attributed to different tissues and organs, taking account of the incidence of fatal cancer and hereditary disease, weighted for the incidence of nonfatal disease and years of life lost (ICRP, 1991).

Table 2. *ICRP f_1 values for members of the public*

Element	Adult	Infant
H, C, I, Cs, S, Mo	1	1
Se	0.8	1
Zn, Tc, Po	0.5	1
Te, Sr*	0.3	0.6
Ba*, Ra*, Pb*	0.2	0.6
Co*, Fe*	0.1	0.6
Sb	0.1	0.2
Ru, Ni, Ag	0.05	0.1
U	0.02	0.04
Zr, Nb	0.01	0.02
Ce, Th, Np, Pu, Am	0.0005	0.005

* Intermediate values for 1, 5, 10, 15 y-old children: Sr, Pb, 0.4; Co, Ra, Ba, 0.3; Fe, 0.2.
f_1 is the fraction of the total ingested that is absorbed to blood.

Table 3. *Committed effective doses from ingestion of radionuclides by adults and infants*

Nuclide	Principal emissions	Half-life (d/yr)	Committed effective dose (Sv Bq^{-1}) Adult	Infant
^{3}H	β	12.3 y	1.8×10^{-11}	6.3×10^{-11}
^{60}Co	β, γ	5.3 y	3.4×10^{-9}	5.4×10^{-8}
^{90}Sr	β	28.5 y	2.8×10^{-8}	2.3×10^{-7}
^{95}Zr	β, γ	64 d	9.6×10^{-10}	8.5×10^{-9}
^{95}Nb	β, γ	35 d	5.9×10^{-10}	4.6×10^{-9}
^{106}Ru	β	368 d	7.0×10^{-9}	8.4×10^{-8}
^{131}I	β, γ	8 d	2.2×10^{-8}	1.8×10^{-7}
^{137}Cs	β	30 y	1.4×10^{-8}	2.1×10^{-8}
^{144}Ce	β	285 d	5.2×10^{-9}	6.6×10^{-8}
^{210}Pb	β	22 y	7.0×10^{-7}	8.1×10^{-6}
^{210}Po	α	138 d	1.2×10^{-6}	2.1×10^{-5}
^{226}Ra	α	1600 y	2.8×10^{-7}	4.7×10^{-6}
^{232}Th	α	1.4×10^{10} y	1.1×10^{-6}	1.4×10^{-5}
^{238}U	α	4.5×10^{9} y	4.5×10^{-8}	3.3×10^{-7}
^{237}Np	α	2.1×10^{6} y	1.1×10^{-7}	2.0×10^{-6}
^{239}Pu	α	2.4×10^{4} y	2.5×10^{-7}	4.2×10^{-6}
^{241}Am	α	433 y	2.1×10^{-7}	3.7×10^{-6}

The committed effective dose is then the sum of all committed equivalent doses multiplied by the appropriate tissue weighting factors. The committed effective dose can be interpreted in terms of risk estimates for whole body exposure. Committed effective dose is applicable to workers and members of the public including children. Table 3 gives values of committed effective dose for the ingestion of a number of radionuclides by adults (20 y) and infants (3 mo).

The least toxic radionuclide, because of its weak β emissions and short biological half-times, is ^{3}H ingested as $^{3}H_2O$ for which an intake of 56 MBq by an adult is equivalent to an external whole body dose of 1 mSv (1.8×10^{-11} Sv Bq^{-1}; Table 3). The probability of different cancer types would be the same for ^{3}H as for external irradiation because of the uniform distribution of ^{3}H in the body. As shown in Table 4, the combined dose to the stomach and colon from ^{3}H accounts for 24% of the committed effective dose, corresponding to the use of tissue weighting factors of 0.12 for each of these regions of the GI tract.

The most toxic radionuclide considered is the α emitter ^{210}Po for which ingestion by an adult of 0.8 MBq is equivalent to an external whole body dose of 1 mSv in terms of total estimated risk. However, the probability of different cancer types would be different. For ^{210}Po, doses to the liver and red bone marrow contribute 25–30% each to the committed effective dose compared with 5% and 12%, respectively, for uniform irradiation. As shown in Table 4, contributions to the committed effective dose from irradiation of the GI tract are smaller than from uniform whole body irradiation. The long-lived α emitting actinide nuclides, ^{232}Th, ^{239}Pu and ^{241}Am, also have relatively high radiotoxicities. Despite their low absorption from the GI tract ($f_1 = 5 \times 10^{-4}$ in adults, 5×10^{-3} in infants; Table 2), the committed effective doses from these nuclides are dominated by doses to the bone surfaces, red bone marrow and liver. Because of their long retention times in these tissues, doses are delivered over an individual's life-time.

As shown in Table 4, examples of radionuclides for which doses to the GI tract contribute a large proportion to the committed effective dose are ^{95}Zr, ^{95}Nb, ^{106}Ru and ^{144}Ce. The importance of doses to the GI tract in these cases is due to a combination of factors. They are all β emitters with short physical half-lives and their absorption from the GI tract is low, particularly for ^{144}Ce ($f_1 = 5 \times 10^{-4}$ in adults, 5×10^{-3} in infants; Table 3). For ^{95}Zr, ^{95}Nb and ^{106}Ru, a large fraction of the absorbed nuclide has a short half-time of retention in the body.

Implications of particle uptake

In general, as discussed above, ingestion of radionuclides in particulate form or contained within particles is likely to lead to lower effective doses than ingestion of more soluble forms although, as illustrated, differences in doses will depend greatly on the radionuclide concerned. However, it is possible that intakes of particulate material could lead to greater doses to regions of the GI tract and also to associated lymphatic tissue. Retention of radioactive materials in the wall of the GI tract has been shown to be a general phenomenon in neonatal animals (see section on new models). Particle uptake in adult animals, by per-

Table 4. *Committed equivalent doses to stomach and colon from ingestion of radionuclides by adults and infants*

	Committed equivalent dose, Sv Bq^{-1}				Total per cent of committed effective dose	
	Stomach		Colon			
Nuclide	Adult	Infant	Adult	Infant	Adult	Infant
^{3}H	1.8×10^{-11}	6.3×10^{-11}	1.8×10^{-11}	6.3×10^{-11}	24	24
^{60}Co	2.6×10^{-9}	5.1×10^{-8}	8.9×10^{-9}	7.7×10^{-8}	42	29
^{90}Sr	9.1×10^{-10}	1.5×10^{-8}	1.3×10^{-8}	1.2×10^{-8}	6	1
^{95}Zr	3.9×10^{-10}	3.5×10^{-9}	5.1×10^{-9}	5.1×10^{-8}	69	76
^{95}Nb	2.9×10^{-10}	2.4×10^{-9}	2.7×10^{-9}	2.4×10^{-8}	61	68
^{106}Ru	3.2×10^{-9}	4.4×10^{-8}	4.6×10^{-8}	5.1×10^{-7}	84	79
^{131}I	3.1×10^{-10}	3.5×10^{-9}	1.2×10^{-10}	2.6×10^{-9}	0.2	0.4
^{137}Cs	1.3×10^{-8}	2.2×10^{-8}	1.5×10^{-8}	3.7×10^{-8}	24	34
^{144}Ce	1.1×10^{-9}	1.4×10^{-8}	4.2×10^{-8}	4.8×10^{-8}	98	89
^{210}Pb	9.1×10^{-8}	2.1×10^{-6}	9.7×10^{-8}	2.2×10^{-6}	3	6
^{210}Po	2.8×10^{-7}	4.6×10^{-6}	3.0×10^{-7}	4.8×10^{-6}	6	5
^{226}Ra	4.1×10^{-8}	5.4×10^{-7}	1.0×10^{-7}	1.2×10^{-6}	6	4
^{232}Th	6.1×10^{-8}	1.7×10^{-6}	8.6×10^{-8}	2.0×10^{-6}	2	3
^{238}U	2.6×10^{-8}	1.3×10^{-7}	5.2×10^{-8}	4.3×10^{-7}	21	20
^{237}Np	8.4×10^{-9}	3.1×10^{-7}	4.0×10^{-8}	6.8×10^{-7}	5	6
^{239}Pu	1.6×10^{-8}	5.6×10^{-7}	4.8×10^{-8}	9.5×10^{-7}	3	2
^{241}Am	1.7×10^{-8}	4.4×10^{-7}	5.3×10^{-8}	8.5×10^{-7}	4	4

sorption or other mechanisms, has been shown to vary considerably according to the species and diet and the particle size and characteristics (O'Hagan, 1990; Jepson et al. 1993; Hodges et al. 1995). Uptake appears to be greatest for hydrophobic particles such as polystyrene. It would appear that the extent of uptake of polystyrene particles of around 1 μm diameter in the intestine of rodents might be about 10^{-5}–10^{-6} of the ingested number of particles. Different mechanisms have been suggested including uptake into villous epithelial cells, uptake by the M cells of Peyer's patches, uptake by macrophages and intracellular uptake. Further studies are required to determine the likely extent of uptake of radioactive particles by such mechanisms in humans and the effect on doses to sensitive cells in the intestinal epithelium and other tissues.

CONCLUSIONS

Radionuclides vary considerably in their distribution and retention within the tissues and organs of the body and in their resulting toxicity. Intakes as radioactive particles, by ingestion or inhalation, may result in a greater proportion of the dose being delivered to the GI tract or lung with lower transfer to other body tissues. For intakes by ingestion, a new dosimetric model is needed which will include considerations of uptake and retention in intestinal tissue for different physicochemical forms of radionuclides, including particles. In determining cancer risks, the available evidence indicates that local irradiation of tissue, as occurs with particles containing nuclides with short-range emissions (e.g. α emitters), is less effective than the same dose delivered more uniformly, as occurs with more soluble chemical forms.

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